

Submission

Impact of microplastics and other toxics on human health

Thank you for inviting the Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG, the College) to make a submission to the *Impact of microplastics and other toxics on human health*.

RANZCOG is the peak professional body for women's health in Australia and New Zealand. The College is responsible for setting clinical standards and overseeing postgraduate education, accreditation, recertification, and continuing professional development for practitioners, including specialist obstetricians, gynaecologists, and GP obstetricians.

Background

Microplastics (MPs) are now ubiquitous across the global environment. Defined as plastic particles less than 5 mm in size, they may be *primary* (manufactured at microscopic scale) or *secondary* (formed through degradation of larger plastic products). Human exposure occurs through inhalation, ingestion, and dermal contact. MPs have been detected in human blood, urine, faeces, breast milk, and sputum, with accumulation documented in organs including the lung, colon, spleen, and placenta.

Endocrine-disrupting chemicals (EDCs), many of which are associated with MPs or plastic production, interfere with hormonal signalling, reproductive function, and systemic metabolic regulation. Evidence indicates that the impacts of MPs and EDCs may begin even before conception, affecting gamete quality and early embryonic development. The processes of reproduction, pregnancy and foetal development are highly sensitive to environmental exposures, especially EDCs, due to tissue remodelling and hormone dependence.¹ The effects on women's health studied thus far have largely been mediated by EDC exposure.

Reproductive and developmental impacts of exposure in males have been associated with:

- Reduced testosterone levels.
- Disruptions to genital development following in utero exposure.
- Possible associations with reduced semen parameters, though findings remain inconsistent.

In females, studies show that:

- MPs within the reproductive tract induce oxidative stress, fibroblast proliferation, and fibrosis.
- Exposure is associated with infertility.
- Micro- and nanoplastics have been identified in placental tissue and meconium.
- Animal studies demonstrate links with fetal growth restriction.
- PFAS exposure is consistently associated with an increased risk of hypertensive disorders of pregnancy.
- EDCs are linked to neurodevelopmental outcomes including ADHD, reduced IQ, and broader executive function deficits.
- Exposure increases the risk of gestational diabetes and childhood obesity.

¹ Schjenken JE, Green ES, Overduin TS, Mah CY, Russell DL, Robertson SA. Endocrine Disruptor Compounds-A Cause of Impaired Immune Tolerance Driving Inflammatory Disorders of Pregnancy? *Front Endocrinol (Lausanne)*. 2021;12:607539.

Beyond fertility and pregnancy outcomes, MPs and EDCs have been implicated in several gynaecological conditions:

- Polycystic ovary syndrome (PCOS): associations with bisphenol A (BPA).
- Endometriosis: links to organochlorine exposures such as pesticides, polychlorinated biphenyls (PCBs), and dioxins.
- Uterine fibroids: associations with phthalate exposure.
- Menopause: PFAS, phthalates, persistent organic pollutants (POPs), and heavy metals have been linked to premature ovarian insufficiency and earlier menopause.
- Ovarian cancer: possible PFAS-related oestrogen receptor modulation and impaired immune surveillance.
- Uterine cancer: biologically plausible links between PFAS exposure and carcinogenesis, particularly in hormonally sensitive individuals.
- Breast cancer: associations with airborne carcinogens including ethylene oxide, nitrogen dioxide, polycyclic aromatic hydrocarbons, benzene, and second-hand smoke.

Fertility

The processes of oocyte production and spermatogenesis are tightly regulated by the hypothalamic pituitary gonadal axis and are highly sensitive to environmental disruption. Exposure to environmental toxins, including heavy metals and EDCs, has been shown to negatively affect both female and male fertility, contributing to reduced fecundability and sperm quality.

Air pollution contains PM2.5 particles and nitrogen dioxide, both of which are associated with lower Anti Müllerian hormone (AMH) levels, a key measure of ovarian reserve. It also contains formaldehyde which is associated with reduced sperm count. BPA, an EDC capable of binding to oestrogen receptors, has similarly been associated with decreased fecundability². Although evidence regarding the direct effects of microplastics on human fertility is still emerging, early findings suggest potential disruption of the hypothalamic pituitary gonadal axis, although robust human data remain limited. In addition, common contributors to female infertility, including fibroids, endometriosis and PCOS, are themselves increasingly associated with exposure to EDCs and air pollutants, compounding concerns regarding environmental impacts on reproductive health.

Gynaecological Conditions

PCOS is a multisystem condition that affects endocrinological, reproductive, cardiometabolic, dermatologic and psychological health³. BPA is the most extensively studied endocrine-disrupting chemical in relation to PCOS, with higher BPA levels consistently associated with increased androgen concentrations, a central feature of PCOS pathogenesis⁴. Importantly, emerging evidence indicates that BPA analogues developed as substitutes after concerns about BPA safety are also associated with PCOS, suggesting that these replacement compounds may not offer a safer alternative^{5,6}.

Fibroids are hormone-dependent tumours, and increasing evidence suggests that EDCs contribute to both their development and progression. Compounds implicated include phthalates, alternative plasticisers, organophosphate esters and phenols. Endometriosis, another highly prevalent gynaecological condition with

² Wang B, Zhou W, Zhu W, Chen L, Wang W, Tian Y, et al. Associations of female exposure to bisphenol A with fecundability: Evidence from a preconception cohort study. *Environ Int.* 2018;117:139-45.

³ Teede HJ, Tay CT, Laven JJE, Dokras A, Moran LJ, Piltonen TT, et al. Recommendations From the 2023 International Evidence-based Guideline for the Assessment and Management of Polycystic Ovary Syndrome. *J Clin Endocrinol Metab.* 2023;108(10):2447-69.

⁴ Kandaraki E, Chatzigeorgiou A, Livadas S, Palioura E, Economou F, Koutsilieris M, et al. Endocrine Disruptors and Polycystic Ovary Syndrome (PCOS): Elevated Serum Levels of Bisphenol A in Women with PCOS. *The Journal of Clinical Endocrinology & Metabolism.* 2011;96(3):E480-E4.

⁵ Zhan W, Tang W, Shen X, Xu H, Zhang J. Exposure to bisphenol A and its analogs and polycystic ovarian syndrome in women of childbearing age: A multicenter case-control study. *Chemosphere.* 2023;313:137463

⁶ Adamovsky O, Groh KJ, Białk-Bielińska A, Escher BI, Beaudouin R, Mora Lagares L, et al. Exploring BPA alternatives – Environmental levels and toxicity review. *Environment International.* 2024;189:108728.

substantial social and economic burden, has similarly been associated with exposure to multiple EDCs and air-pollution-related toxicants, including phthalates, dioxins and PCBs⁷.

EDCs, including phthalates, PFAS and POPs, have been associated with earlier onset of menopause and premature ovarian insufficiency. These changes to ovarian physiology are known to increase lifelong risks of cardiovascular disease and osteoporosis. The contribution of EDCs to the pathogenesis of fibroids and endometriosis is also substantial. Hunt et al. estimate that exposure to diphenyldichloroethene and phthalates costs the European Union nearly 1.5 billion euros annually, reflecting the significant disease burden.⁸

Pregnancy

There is growing awareness of the importance of the first one thousand days, the period from conception to the end of the second year of life when the developing human is exquisitely sensitive to the environment.⁹ Physiological changes in pregnancy, such as delayed gastric emptying and increased minute ventilation, heighten both the severity and duration of toxins and microplastics exposure. These adaptations also increase susceptibility to harm from environmental toxins, as pregnancy places the body in a borderline pathological state with reduced capacity to buffer external stressors.

Physiological changes in pregnancy, including delayed gastric emptying and increased minute ventilation, extend and intensify exposure to toxins and microplastics.¹⁰ These adaptations also heighten vulnerability to environmental harms, as the altered physiological state of pregnancy reduces the body's ability to buffer external stressors.¹¹

Environmental pollutants, including EDCs and micro- and nanoplastics (MNPs), can impair placental function, affecting maternal health and indirectly disrupting fetal development. Many of these substances can cross the placenta and exert direct effects on the fetus, and exposure may continue postpartum through the transfer of EDCs into breastmilk.¹²

A growing body of evidence demonstrates that environmental toxins have substantial impacts across the reproductive continuum, including miscarriage, preterm birth, hypertensive disorders of pregnancy, gestational diabetes, perinatal mortality and long-term childhood development. Animal, in vitro and observational human studies have shown associations between elevated BPA levels and miscarriage, with dose-dependent relationships also reported for exposures to PCBs, DDT and phthalates.¹³ Recurrent pregnancy loss has additionally been linked to male occupational toxin exposure through mechanisms involving sperm DNA fragmentation.^{14,15}

⁷ Cano-Sancho G, Ploteau S, Matta K, Adoamnei E, Louis GB, Mendiola J, et al. Human epidemiological evidence about the associations between exposure to organochlorine chemicals and endometriosis: Systematic review and meta-analysis. *Environment International*. 2019;123:209-23.

⁸ Hunt PA, Sathyanarayana S, Fowler PA, Trasande L. Female Reproductive Disorders, Diseases, and Costs of Exposure to Endocrine Disrupting Chemicals in the European Union. *J Clin Endocrinol Metab*. 2016;101(4):1562-70

⁹ Barnden, K. (2022, June 2). *A GP guide to environmental impacts on pregnancy*. AusDoc. <https://www.ausdoc.com.au/therapy-update/gp-guide-environmental-impacts-pregnancy/>

¹⁰ Vinnars M-T, Bixo M, Damdimopoulou P. Pregnancy-related maternal physiological adaptations and fetal chemical exposure. *Molecular and Cellular Endocrinology*. 2023;578:112064.

¹¹ Varshavsky J, Smith A, Wang A, Hom E, Izano M, Huang H, et al. Heightened susceptibility: A review of how pregnancy and chemical exposures influence maternal health. *Reprod Toxicol*. 2020;92:14-56.

¹² Gingrich J, Ticiani E, Veiga-Lopez A. Placenta Disrupted: Endocrine Disrupting Chemicals and Pregnancy. *Trends Endocrinol Metab*. 2020;31(7):508-24.

¹³ Krieg SA, Shahine LK, Lathi RB. Environmental exposure to endocrine-disrupting chemicals and miscarriage. *Fertility and Sterility*. 2016;106(4):941-7.

¹⁴ Ruixue W, Hongli Z, Zhihong Z, Rulin D, Dongfeng G, Ruizhi L. The impact of semen quality, occupational exposure to environmental factors and lifestyle on recurrent pregnancy loss. *J Assist Reprod Genet*. 2013;30(11):1513-8.

¹⁵ Suker A, Li Y, Robson D, Marren A, Group tACCEPoTE. Australasian recurrent pregnancy loss clinical management guideline 2024, part II. *Australian and New Zealand Journal of Obstetrics and Gynaecology*. 2024;64(5):445-58.

Preterm birth remains the leading cause of death and disability in children under five years in the developed world and, in severe cases, can result in cerebral palsy, sensory impairment, chronic lung disease and developmental delay^{16,17}. Even late preterm and early term birth are associated with behavioural and learning difficulties, indicating a broad spectrum of morbidity. While preterm birth has multiple causes, including conditions such as pre-eclampsia, environmental exposures represent a modifiable risk factor. Strong evidence supports a role for air pollution, including exposures occurring during the periconception period when pregnancy may not yet be recognised, in elevating the risk of preterm birth^{18,19}. High-quality studies also demonstrate that EDCs such as PFAS and phthalates increase preterm birth rates.^{20,21}

Hypertensive disorders of pregnancy, including pre-eclampsia, are common obstetric complications and the second leading cause of maternal mortality globally.²² Exposure to traffic-related ambient air pollution and to PFAS, including legacy PFAS compounds, has been associated with higher rates of pregnancy-related hypertension.^{23,24} Gestational diabetes similarly poses significant risks, contributing to hypertensive disorders, emergency caesarean section, neonatal hypoglycaemia and long-term metabolic dysfunction in offspring due to altered in-utero glucose and lipid metabolism.²⁵ Particulate matter exposure from air pollution is associated with increased rates of gestational diabetes, and prenatal exposure to BPA, phthalates, POPs and PFAS has been linked to higher rates of childhood obesity and early-life metabolic syndrome.^{26,27,28}

In-utero and early-childhood exposure to environmental toxins is also strongly implicated in paediatric neurodevelopmental outcomes. There is robust evidence linking air pollution, EDCs, heavy metals and pesticides to increased rates of autism spectrum disorder, ADHD, reduced IQ and broader cognitive impairment.²⁹ Although data on the impact of environmental exposures on perinatal mortality and second- or third-trimester pregnancy loss remain limited, small observational studies show correlations between air pollution, certain occupational exposures and fetal death in utero.^{30,31} Moreover, as demonstrated above, environmental toxins elevate the risk of pre-eclampsia and preterm birth, both of which are major contributors to perinatal mortality. These pregnancy pathologies, including gestational diabetes and hypertensive disorders, also increase the long-term cardiovascular and metabolic disease burden for affected mothers.

¹⁶ Newnham JP, Vernon BA, Ward SV, Brown K, Lehner C, Kane SC, et al. Reducing rates of preterm and early-term singleton births safely in Australia: results of the national prevention programme. *The Lancet Obstetrics, Gynaecology, & Women's Health*. 2025;1(4):e291-e301.

¹⁷ Saigal S, Doyle LW. An overview of mortality and sequelae of preterm birth from infancy to adulthood. *The Lancet*. 2008;371(9608):261-9.

¹⁸ Bekkar B, Pacheco S, Basu R, DeNicola N. Association of Air Pollution and Heat Exposure With Preterm Birth, Low Birth Weight, and Stillbirth in the US: A Systematic Review. *JAMA Network Open*. 2020;3(6):e208243-e.

¹⁹ Blanc N, Liao J, Gilliland F, Zhang JJ, Berhane K, Huang G, et al. A systematic review of evidence for maternal preconception exposure to outdoor air pollution on Children's health. *Environ Pollut*. 2023;318:120850

²⁰ Wu Y, Wang J, Wei Y, Chen J, Kang L, Long C, et al. Maternal exposure to endocrine disrupting chemicals (EDCs) and preterm birth: A systematic review, meta-analysis, and meta-regression analysis. *Environ Pollut*. 2022;292(Pt A):118264.

²¹ Gui SY, Chen YN, Wu KJ, Liu W, Wang WJ, Liang HR, et al. Association Between Exposure to Per- and Polyfluoroalkyl Substances and Birth Outcomes: A Systematic Review and Meta-Analysis. *Front Public Health*. 2022;10:855348.

²² Kinshella M-LW, Pickerill K, Prasad S, Campbell O, Devji J, Lopes LV, et al. Towards the Development of a Conceptual Framework of the Determinants of Pre-Eclampsia: A Hierarchical Systematic Review of Social Determinants. *BJOG: An International Journal of Obstetrics & Gynaecology*. n/a(n/a).

²³ NTP monograph on the systematic review of traffic-related air pollution and hypertensive disorders of pregnancy. *NTP Monogr*. 2019(7).

²⁴ Hu C-Y, Qiao J-C, Gui S-Y, Xu K-X, Dzhambov AM, Zhang X-J. Perfluoroalkyl and polyfluoroalkyl substances and hypertensive disorders of pregnancy: A systematic review and meta-analysis. *Environmental Research*. 2023;231:116064.

²⁵ Sheiner E. Gestational Diabetes Mellitus: Long-Term Consequences for the Mother and Child Grand Challenge: How to Move on Towards Secondary Prevention? *Front Clin Diabetes Healthc*. 2020;1:546256.

²⁶ Braun JM. Early-life exposure to EDCs: role in childhood obesity and neurodevelopment. *Nat Rev Endocrinol*. 2017;13(3):161-73.

²⁷ Shin MW, Kim SH. Hidden link between endocrine-disrupting chemicals and pediatric obesity. *Clin Exp Pediatr*. 2025;68(3):199-222.

²⁸ Yan D, Jiao Y, Yan H, Liu T, Yan H, Yuan J. Endocrine-disrupting chemicals and the risk of gestational diabetes mellitus: a systematic review and meta-analysis. *Environ Health*. 2022;21(1):53.

²⁹ Bennett D, Bellinger DC, Birnbaum LS, Bradman A, Chen A, Cory-Slechta DA, et al. Project TENDR: Targeting Environmental Neuro-Developmental Risks The TENDR Consensus Statement. *Environ Health Perspect*. 2016;124(7):A118-22.

³⁰ Ooka T, Horiuchi S, Shinohara R, Kojima R, Akiyama Y, Miyake K, et al. Association between Maternal Exposure to Chemicals during Pregnancy and the Risk of Foetal Death: The Japan Environment and Children's Study. *International Journal of Environmental Research and Public Health*. 2021;18(22):11748.

³¹ Tong M, Lin W, Liu H, Gong J, Zhang J, Xue T. Gestational age modifies the association between exposure to fine particles and fetal death: findings from a nationwide epidemiological study in the contiguous United States. *Environmental Health*. 2023;22(1):65.

Epigenetics

Epigenetics describes the ability for environmental exposures to change the expression of DNA and therefore phenotype and susceptibility to pathology. These epigenetic modifications can be inherited by subsequent generations, meaning that harmful environmental exposures have the potential to cause ongoing morbidity long after the initial exposure has ended. Multiple endocrine-disrupting chemicals, including PFAS, BPA and phthalates, have been implicated in such epigenetic alterations.

Microplastics have been identified in placental tissue, amniotic fluid and umbilical cord blood, raising concern about early developmental exposure. Although research on clinical outcomes remains limited to small observational studies, emerging evidence suggests that micro- and nanoplastics can accumulate within ovarian tissue, contributing to reduced oocyte quality and anovulatory cycles. Associations have also been reported between microplastic exposure and increased risks of preterm birth and intrauterine growth restriction, highlighting the need for more comprehensive investigation into the reproductive and developmental impacts of these contaminants.^{32,33,34}

The Role of Health Practitioners

RANZCOG recognises that health practitioners and those developing educational resources must aim to strike an appropriate balance when discussing environmental risks with patients. Communication should alert individuals to potential harms in a way that is accurate but not alarmist, while acknowledging that many environmental chemicals are ubiquitous and cannot be completely avoided. Emphasis should be placed on simple, achievable steps to reduce exposure and thereby minimise potential harm. Clinicians also need the capacity to recognise patients with high-risk exposures, whether occupational, domestic or community-based, and to advocate on their behalf where appropriate, for example with employers, landlords or local councils.

Obstetricians and gynaecologists are particularly well-positioned to support clinical and public health efforts to identify and mitigate environmental risks.^{35,36} RANZCOG supports its members to integrate environmental health into routine reproductive care through screening, counselling, advocacy and policy engagement. However, O&G specialists cannot carry this responsibility alone. They require coordinated support through government policy, robust regulatory frameworks and widespread public education.³⁷

Patient-centred strategies can meaningfully reduce body burdens of toxic chemicals, and RANZCOG agrees that health practitioners have a duty of care to discuss environmental risks with patients.³⁸ To fulfil this role effectively, clinicians must be supported with appropriate training, evidence-based guidance and practical resources. Nonetheless, responsibility cannot rest solely with individuals, particularly given that those most vulnerable to environmental exposures often have the least agency to change their circumstances. Legislative

³² Jochum M, Garcia M, Hammerquist A, Howell J, Stanford M, Liu R, et al. Elevated Micro- and Nanoplastics Detected in Preterm Human Placentae. *Res Sq*. 2025.

³³ Panneerselvam D, Murugesan A, Raveendran SK, Kumar JS, Venkataraman P. Examining the hidden dangers: Understanding how microplastics affect pregnancy. *European Journal of Obstetrics & Gynecology and Reproductive Biology*. 2025;304:53-62.

³⁴ Ali-Hassanzadeh M, Arefinia N, Ghoreishi ZA, Askarpour H, Mashayekhi-Sardoo H. The effects of exposure to microplastics on female reproductive health and pregnancy outcomes: A systematic review and meta-analysis. *Reprod Toxicol*. 2025;135:108932.

³⁵ DeNicola N, Zhang J, Hasenburger A, et al. FIGO committee opinion: Environmental drivers of gynecologic and reproductive health. *Int J Gynecol Obstet*. 2025; 171: 1008-1021. doi:10.1002/ijgo.70547

³⁶ Stotland NE, Sutton P, Trowbridge J, Atchley DS, Conry J, Trasande L, Gerbert B, Charlesworth A, Woodruff TJ. Counseling patients on preventing prenatal environmental exposures—a mixed-methods study of obstetricians. *PloS one*. 2014 Jun 25;9(6):e98771.

³⁷ Sutton P, Woodruff TJ, Perron J, Stotland N, Conry JA, Miller MD, Giudice LC. Toxic environmental chemicals: the role of reproductive health professionals in preventing harmful exposures. *American journal of obstetrics and gynecology*. 2012 Sep 1;207(3):164-73.

³⁸ Laferriere, Kathryn & Crighton, Eric. (2016). "During pregnancy would have been a good time to get that information": mothers' concerns and information needs regarding environmental health risks to their children 1. *International Journal of Health Promotion and Education*. 55. 1-10. 10.1080/14635240.2016.1242376.

and regulatory measures are therefore essential to limit the production and distribution of harmful environmental toxins and to ensure the public is adequately informed about potential risks.³⁹

Summary

The evidence clearly demonstrates that microplastics and other environmental toxins pose significant and potentially multigenerational harms to reproductive, obstetric and broader population health. RANZCOG submits that coordinated, system-level action is urgently required to reduce exposure. This must include comprehensive public education on health risks and practical mitigation strategies, targeted training and resources for health practitioners, and strengthened regulatory measures to limit the production, distribution and release of harmful contaminants.

Individual behaviour change cannot address a problem of this scale or complexity. RANZCOG therefore calls for immediate and decisive government leadership, including stronger regulation, mandatory industry accountability and sustained public health communication. Protecting reproductive and developmental health requires treating environmental contaminants as a critical public health priority and acting now to safeguard the wellbeing of current and future generations.

RANZCOG acknowledges with thanks, the contributions of the Environmental and Sustainability Committee for this submission.

Yours sincerely,

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President

³⁹ American College of Obstetricians and Gynecologists. (2021). *Reducing prenatal exposure to toxic environmental agents* (Committee Opinion No. 832). <https://www.acog.org/clinical/clinical-guidance/committee-opinion/articles/2021/07/reducing-prenatal-exposure-to-toxic-environmental-agents>

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